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Prairie Dogs, Pesticides, and Protected Species: Concerns for Anticoagulant Use in a Sensitive Ecosystem

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ABSTRACT: The black-tailed prairie dog occupies an estimated 2.4 million acres in the western U.S and is considered to be a keystone species of the Great Plains due to its influence on biological diversity and ecosystem function. Over 200 vertebrate species are known to associate with prairie dog colonies, and there is documentation that at least 9 species exhibit dependence on prairie dogs either for habitat and shelter or as a prey species. Unlike many other burrowing mammals, the prairie dog relies on an open burrow system that results in a significant amount of time spent above ground, rendering them more easily accessible to predatory species. Many species that use the prairie dog as a food source are protected under the Endangered Species Act, the Migratory Bird Treaty Act, and the Bald and Golden Eagle Protection Act, and include the black-footed ferret, both species of eagle, and several species of raptors. The registration and use of the anticoagulant rodenticides chlorophacinone and diphacinone for prairie dog control presents risks to these protected species due to the potential for secondary poisoning. Anticoagulant rodenticides have caused secondary poisoning in laboratory studies and have been responsible for mortality incidents in the field. Since geographically limited registrations began in 2005, a limited number of such incidents associated with prairie dog control have been documented, and these elicit concern for the more widespread use of these rodenticides that would accompany registrations covering the entire range of the black-tailed prairie dog.

KEY WORDS: anticoagulants, black-tailed prairie dog, chlorophacinone, *Cynomys ludovicianus*, diphacinone, Endangered Species Act, Migratory Bird Treaty Act, nontarget species, rodenticides

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INTRODUCTION

The black-tailed prairie dog (*Cynomys ludovicianus*) occupies an estimated 2.4 million acres of grassland habitat throughout the midwestern United States. Though this represents less than 3% of habitat once occupied within its historic range, prairie dog populations have been on the rise since the 1960s (USFWS 2009a). This increasing population trend was a contributing factor in the recent finding by the U.S. Fish and Wildlife Service (USFWS) that listing the black-tailed prairie dog as threatened or endangered is not warranted at this time (USFWS 2009a). However, the influence of prairie dog colonies extends beyond the species. Many ecologists consider the prairie dog a keystone species, defined as having an overall effect on ecosystem structure or function that is disproportionately large relative to its abundance (Kotliar et al. 1999, Miller et al. 2000, Power et al. 1996). Grazing and burrowing activities by prairie dogs have a large effect on vegetative structure and soil mixing, creating structural complexity that leads to altered species composition in occupied habitats. Over 200 species have been associated with prairie dog colonies, and 9 species have been determined to be dependant on these colonies, meaning the species would decline or disappear at a local or landscape scale if prairie dogs were eliminated (Kotliar et al. 1999). Several of these species have experienced population declines significant enough to warrant special protection or additional study (Mulhern and Powell 1993).

Many of the same features that make prairie dog colonies essential for conservation have also led to their consideration as a pest species. The agricultural community is concerned about depletion of vegetation, soil erosion, and the potential hazard to livestock from burrows. Consequently, prairie dogs have been viewed as threats to farming and ranching. Active removal of colonies via widespread poisoning programs in the first half of the 20th century was a major contributing factor to population declines (Forrest and Luchsinger 2006). At present, removal of colonies via poisoning and other methods continues on a localized scale, where prairie dogs may compete with domestic livestock for forage, interfere with agriculture, threaten humans via disease transmission, or interfere with commercial development of land (Forrest and Luchsinger 2006). Until recently, the most common toxicants used in the control of prairie dogs have been zinc phosphide (2% on oats or grain pellet formulations) and burrow fumigants (aluminum phosphide and incendiary gas cartridges) (Witmer and Fagerstone 2003). However, in 2005 the first registrations were granted for the use of anticoagulant rodenticides to control black-tailed prairie dogs. These registrations have led to concerns regarding the impact of these pesticides on species associated with the colonies, many of which are protected by state or federal conservation laws such as the Endangered Species Act (ESA), Migratory Bird Treaty Act (MBTA), and the Bald and Golden Eagle Protection Act (BGEPA).

SECONDARY EFFECTS OF ANTICOAGULANT RODENTICIDES

Two anticoagulant rodenticides, chlorophacinone (trade name Rozol® Prairie Dog Bait, 0.005% a.i.) and diphacinone (trade name Kaput®-D Prairie Dog Bait, 0.0025% a.i.), have been granted registrations for black-tailed control of prairie dogs by the U.S. Environmental Protection Agency (USEPA). Rozol® is registered under Section 3 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) for use throughout the range of the black-tailed prairie dog (Colorado, Kansas, Montana, Nebraska, New Mexico, North Dakota, Oklahoma, South Dakota, Texas, and Wyoming) with the exception of Arizona, where the species has recently been reintroduced for conservation purposes. Kaput®-D is registered under Section 24c of FIFRA in Colorado, Kansas, Nebraska, Texas, and Wyoming, with a pending request to amend the Kaput® Field Rodent Bait B (0.0025% a.i.) registration to include use on black-tailed prairie dogs throughout their range. Both rodenticides are approved for one or two 2-oz. applications to be placed 6 inches down prairie dog burrows between October 1 and March 15.

Anticoagulant rodenticides cause toxicity by disrupting the synthesis of clotting factors through antagonism of vitamin K (Ecobichon 2001). Exposure can result in capillary damage and hemorrhage leading to death. The risk to secondary consumers from anticoagulants is higher than from historical choices for prairie dog control such as zinc phosphide, due to their ability to persist in tissue of target organisms (Erickson and Urban 2004, Matschke et al. 1992). Secondary exposure may occur when predators feed on primary consumers that have consumed either sublethal or lethal concentrations of these pesticides. In laboratory studies, predators and scavengers fed diphacinone- or chlorophacinone-laced prey suffered mortality and sublethal effects such as external bleeding, internal hematoma, and increased clotting time (Massey et al. 1997, Mendenhall and Pank 1980, Radvanyi et al. 1988, Savarie et al. 1979). These sublethal effects may compromise the fitness of individuals by making them more susceptible to other stressors and hasten death from other causes, though this relationship is difficult to measure in a field setting. As such, most risk assessments focus on mortality and dismiss the potential for adverse impacts to individuals or populations resulting from sublethal effects (e.g., Silberhorn et al. 2003) or discuss them but don't consider them in the final analysis (Erickson and Urban 2004). Those that incorporate sublethal effects into their risk analysis can be limited in their conclusions by the narrow scope of available data beyond acute mortality, but they describe and recognize their potential to affect survival (Eisemann and Swift 2006).

Both chlorophacinone and diphacinone are classified as "first-generation" anticoagulants, which are less acutely toxic than "second-generation" anticoagulants (e.g., brodifacoum) and generally require multiple feedings to kill target organisms. Because there is a delay between exposure and death that generally lasts 5-10 days, primary consumers may continue to feed on available bait and accumulate large loads of anticoagulants that exceed the lethal threshold by the time of death (Erickson

and Urban 2004). As anticoagulants persist in body tissues of poisoned individuals for periods of weeks for first-generation anticoagulants versus hours for non-anticoagulant rodenticides, these residues are likely to accumulate in prey that continue to ingest bait and remain available to nontarget species (Eason et al. 2008). Availability of prairie dogs to secondary consumers is facilitated by their tendency to spend time above ground. Prairie dogs spend a significant amount of time above ground and when subjected to poisoning, may wander around on the surface, becoming increasingly debilitated until death. Anecdotal reports from applicators verify that dead and dying prairie dogs are often found above ground and have been reported to behave as if "intoxicated" (S. Larson, U.S. Fish and Wildlife Service, pers. commun.). An efficacy study of diphacinone observed sick and lethargic black-tailed prairie dogs above ground following treatment (Bruening 2007). Toxicant-exposed organisms can be more susceptible to predation due to changes in behavior that render them more conspicuous and/or less able to adaptively respond to the presence of a predator (Hunt et al. 1992, Relyea and Hoverman 2006). Given these effects, poisoned prairie dogs may be particularly vulnerable to capture by secondary consumers.

A particularly large number of carcasses were available for both predation and scavenging following an illegal application of Rozol® on 160 tribal acres in South Dakota in 2005. Two weeks after the application, over 50 dead, dying, and scavenged prairie dogs were found (B. Prieksat, U.S. Fish and Wildlife Service, pers. commun.). On a follow-up visit 4 weeks after application, it was noted that tribal applicators retrieved an additional 400-500 prairie dogs above ground from the Rozol®-treated site. While this application represents a misuse of Rozol®, it provided data regarding the tendency of at least a portion of the prairie dogs poisoned with chlorophacinone to die above ground. While increased frequency of carcass search and burial has been proposed as a means to reduce the availability of contaminated prey, not all carcasses are likely to be found or can be removed rapidly enough to prevent exposure to nontarget species. In addition, reports from applicators indicate that current label directions are not always followed with regard to carcass searches, carcass burial, and in-burrow application (S. Larson, U.S. Fish and Wildlife Service, pers. commun.; Williams 2009).

The toxicity of anticoagulant-treated prairie dogs to secondary predators has been studied only in domestic ferrets (*Mustela putorius*) as a surrogate for the endangered black-footed ferret (*M. nigripes*) (Fisher and Timm 1987). Significant mortality occurred in ferrets consuming prairie dogs fed bait containing 0.0025% chlorophacinone, half the concentration of the currently registered product. Based on this study, the authors recommended against the use of chlorophacinone on prairie dogs due to the potential secondary hazard. Other studies of secondary toxicity, including those cited above, were performed by feeding consumers prey species substantially smaller than prairie dogs, including rats, voles, and mice. Carcass residue concentrations measured in these species from studies using 0.005% bait were

0.45 and 0.47 mg a.i./kg-bw for rats, 1.58 and 3.2 mg a.i./kg-bw for voles, and 5.8 mg a.i./kg-bw for mice (Ahmed et al. 1996, Askham and Poché 1991, Baroch 1997, Joermann 1998, Primus et al. 2001). The mean whole body average corrected wet weight concentrations for prairie dogs fed Rozol® to mortality was 1.03 mg a.i./kg-bw in a study submitted to EPA by the registrant (Shelby and Grable 2010). Based on these carcass concentrations, and average body weight of prey species, a single prairie dog carcass will carry a substantially larger dose of poison to predatory or scavenging wildlife. Thus, nontarget wildlife would need to feed on a smaller number of poisoned prairie dogs to achieve toxicity associated with consumption of other prey species.

WILDLIFE PROTECTION LAWS

The MBTA, BGEPA, and ESA protect wildlife from injury or harm resulting from human activities, including pesticide use. In administering these laws, the USFWS, and in the case of the ESA, the National Marine Fisheries Service (NMFS), advise federal and state agencies, private landowners, and organizations of ways in which to minimize the adverse effects of rodenticides upon protected species. To date, there has been no comprehensive assessment of the effects upon protected species of any currently registered rodenticide, including chlorophacinone or diphacinone (Golden 2007).

Take of threatened or endangered wildlife, including death, or injury that impacts essential behavioral patterns, including breeding, feeding, or sheltering, is prohibited under the ESA. Federal agencies are required to determine if actions they authorize, fund, or carry out will result in take of threatened or endangered species and ultimately jeopardize their continued existence. When an action by a federal agency may result in take, consultation with USFWS or NMFS is required. As a result of one such consultation, the USFWS completed a Biological Opinion on 16 vertebrate control agents including chlorophacinone and diphacinone in 1993 (USFWS 1993). At that time, the registered uses for these anticoagulants did not include prairie dogs. The 1993 Biological Opinion determined that the registered uses for chlorophacinone and diphacinone would jeopardize the continued existence of numerous species listed under the ESA, including those potentially exposed by secondary poisoning. Adding prairie dog control to the list of registered uses of these pesticides would likely increase the number of adversely affected species. A recent assessment by the USEPA determined that 21 federally endangered and threatened species were likely to be adversely affected by chlorophacinone use on black-tailed prairie dogs (Shelby and Grable 2010). A full assessment of the potential effects to their populations is pending. Of particular concern are effects to the black-footed ferret, which is highly dependent upon black-tailed prairie dogs, both for food and for the utilization of their burrows. In November 2008, the USFWS issued a 5-Year Review of the ferret, citing the poisoning of prairie dogs as a major factor in the decline of ferrets, through both decline of prairie dogs and inadvertent poisoning of ferrets (USFWS 2008a). The report recommends that federal agencies more fully embrace responsibilities under the ESA to

restore and manage viable prairie dog complexes to support ferret recovery, and specifically cites the need for the USEPA to re-address the use of anticoagulants for control of prairie dogs.

The MBTA prohibits the take of migratory birds, including mortality resulting from exposure to pesticides registered under FIFRA [*U.S. v. Corbin Farm Services*, 444 F. Supp. 510 (1978)]. Migratory raptors are especially susceptible to secondary poisoning from anticoagulant use due to their propensity to feed in prairie dog colonies. The golden eagle (*Aquila chrysaetos*), ferruginous hawk (*Buteo regalis*), and burrowing owl (*Athene cunicularia*) have high association with prairie dog colonies and are among 9 species with documented dependence on these ecosystems (Kotliar et al. 1999, Seery and Matiatos 2000). All three of these raptor species have been identified as USFWS Species of Conservation Concern, defined as species that are likely to become candidates for listing under the ESA without additional conservation action (USFWS 2008b). Bald eagles (*Haliaeetus leucocephalus*) and golden eagles are additionally protected under the BGEPA. While the bald eagle was recently removed from the endangered species list in most of its range (72 FR 37345), golden eagle populations appear to be experiencing declines throughout most of their range, such that populations may not be able to withstand additional loss of individuals (USFWS 2009b). Bald eagles, in turn, may be particularly vulnerable to residue exposure, as they are kleptoparasitic associates of ferruginous hawks, a superior predator of terrestrial prey highly associated with prairie dog colonies (Jorde and Lingle 1988).

KNOWN INCIDENTS INVOLVING NONTARGET SPECIES

Documented mortalities have been reported in badgers (*Taxidea taxus*) and a bald eagle from secondary poisoning following the legal application of chlorophacinone in prairie dog colonies (CAHFS 2009, USFWS 2007). Other incidents have confirmed anticipated exposure pathways between treated prairie dogs and predators or scavengers feeding in colonies. In one incident, chlorophacinone residues were coupled with the presence of prairie dog hair in the gut of a carcass analyzed from a die-off involving a great horned owl (*Bubo virginianus*), golden eagle, and ferruginous hawks near the site of a Rozol® application (USFWS 2009c). In Bruening's (2007) study of diphacinone efficacy, the researchers observed a bald eagle flying off the treatment plot with a prairie dog in its talons, and noted the presence of other predatory and scavenging avian species (e.g., red-tailed hawks *Buteo jamaicensis*, golden eagles, black-billed magpies *Pica hudsonia*, and turkey vultures *Cathartes aura*) multiple times on or near the study site.

The actual number of non-target species impacted is likely much greater, and it is anticipated to rise with the use of anticoagulant rodenticides on prairie dogs over a greater geographic area. However, the ability to verify impacts in the field to non-target species is quite limited. In order to document a pesticide-related mortality, a carcass must be observed, reported, collected, and chemically analyzed while still relatively fresh. Carcass-

detection studies have found that even when searches are performed on known carcasses, a significant percentage will never be found due to scavenging, location in remote, inaccessible areas, or size or coloration that renders the carcass inconspicuous (Vyas 1999). In the case of anticoagulants, the delayed toxicity can temporally or geographically distance the carcass from the application (Colvin et al. 1988). Therefore, only a very small percentage of animals that die from secondary poisoning will be located and incident reports are likely to represent only a fraction of the actual mortality for any given pesticide (Vyas 1999).

CONCLUSION

Prairie dog colonies are unique ecosystems that support numerous species of wildlife. While non-anticoagulant toxicants have historically been one of the methods used to control prairie dogs, the recent registrations of chlorophacinone and diphacinone for this purpose present new risk to species protected under the MBTA, BGEPA, and ESA. Though species are managed at the population level under these statutes, regulation and enforcement often occurs at the level of the individual. Thus, the take of a single individual of a protected species as the result of rodenticide exposure can amount to a violation of these acts. The few individual mortalities verified from this new pattern of use likely represent a greater impact to predatory and scavenging species than can be documented by passive or even active carcass retrieval, and they indicate that either existing risk assessments for chlorophacinone and diphacinone are not adequately predicting risks for this system, or that current risk management is insufficient. While requirements that are designed to remove treated prey from the food web have been suggested as additions to the labeled instructions for use of these pesticides, we do not believe that this approach will adequately remove risk to protected species. Carcass search and retrieval by applicators is unlikely to exceed the rate that can be achieved by predators and scavengers, and mandating an increased frequency of searches runs the risk of incurring greater noncompliance and providing the false perception of protection. Under Section 3(c)5 of FIFRA, a pesticide is eligible for registration "when used in accordance with widespread and commonly recognized practice, it will not generally cause unreasonable adverse effects to the environment". Take of protected species is a logical interpretation of what constitutes an unreasonable adverse effect. We believe that mortality and exposure incidents should be considered alongside reports of label noncompliance regarding carcass removal and burial when determining common practice. These should be integrated into a more accurate analysis of ecological effects for the assessment of eligibility for registration.

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